since Schultze and Langard have observed that the reflexes may be exaggerated to the highest degree in a condition of mental excitement and in chronic diseases, particularly in phthisis.

6. *The Various Forms of Disease Observed in Trauma.*—As on former occasions he earnestly advises that these nervous disturbances should not be accepted as exclusively due to trauma, but that etiological factors should be considered. In the twenty cases there was one with typical chorea minor; four with Ménière's disease, and one with sciatica. In others, the symptoms could be ascribed to pulmonary tuberculosis or to arterio-sclerosis with cardiac hypertrophy.

7. *Simulation and Exaggeration.*—He points out the great difficulties, and analyzes several of the most doubtful symptoms, and again emphasizes, that in general the best means for determining the correctness of the patient's statements is by accurate and assiduous methods of observation. Among the twenty cases there were two of simulation; and in six patients the symptoms were in a great measure highly exaggerated.

8. He does not consider the prognosis unfavorable, and discourages the impression that the diagnosis of "traumatic neurosis" is synonymous with a death-warrant. The essay closes with seven well-selected and instructive histories (Centrbl. f. klin. Med., No. 30, 1892).

W. M. L.

TABES AND PARESIS.

In "Médicine moderne," June 16, 1892, there is an abstract of Rendu's paper with the foregoing title. The clinical association of tabes and paresis is now beyond dispute, though attempts have been made to prove some clinical differences between tabetic symptoms that are epiphenomena of paresis and those of true ataxia, before cerebral symptoms appear. These fine distinctions vanish before an analysis of facts. In two cases cited in the paper, the symptoms are those of classic ataxia, up to the moment that evidences of diffuse encephalitis appeared. According to Raymond, true tabes, as we know it, does not always confine itself exclusively to lesions of the posterior part of the cord, but extends into the motor cells and lateral tracts. Thus it would appear there is often in ataxia a systemic disease, a tendency to diffusion of the sclerotic process. Because certain isolated symptoms appear in subjects attacked by true ataxia and general paresis, there is no reason to seek other diagnosis and invent a special type of disease. At present it is impossible to distinguish between syphilitic and non-syphilitic ataxia. Theoretically, as Ballet says, there are probably different kinds of locomotor ataxia; but their differential characteristics are as yet purely hypothetical. Certain etiological factors that produce tabes and paresis are alike, namely, alcohol and syphilis. Admitting that heredity plays a part as important as these, is but to emphasize another point in common. Upon the question of morbid anatomy and extension of morbid processes opinions are divided. On histological grounds Ballet and Joffroy reject the idea of any identity of tabes and paresis. Differences of opinion in this matter are more apparent than real, for when these abnormities are both present, the symptoms are complex, involving the cerebral and spinal vessels, the neuroglia and nerve cells. The question at issue is whether the initial lesion begins in the vessels, the axis cylinder or the nerve cells. Raymond considers the vascular lesion predominant. Ballet finds that the beginning of the morbid process is in the neuroglia. And Joffroy inclines to the opinion that the cerebral cells are primarily at fault in paresis, the interstitial sclerosis being secondary. Naturally there is some disparity in conclusions based upon premises so diverse. Raymond says, endarteritis exists in the cord as well as the brain and sclerosis follows in consequence, diffuse in the brain and systemic in the cord, the morbid process being identical because the disease is the same though manifested in different regions. Ballet, on the contrary, considers locomotor ataxia a myelitis of peri-cylindrical origin, neuroglia and spinal vessels being implicated secondarily. Paresis, on the contrary, is a diffuse encephalitis of probable vascular origin. The fundamental abnormity is, therefore, distinctly different, and the two diseases bear no relation other than chance association. Joffroy looks upon the cell as primarily imperfect. General paresis and locomotor ataxia are parenchymatous inflammations of the nervous system. This community of cause might have been expected to throw some light upon the identity of the morbid process. Joffroy, on the contrary, rejects the idea, saying that ataxia is a systemic lesion while paresis is a diffuse lesion. The two conditions may be associated, but this is mere coincidence. At the present stage of knowledge it would be difficult to prove that locomotor ataxia begins in the axis cylinder, and that paresis is the outcome of abnormal neuroglia and abnormal blood-vessels. Personally, Rendu, the author of the paper, inclines to the belief that in visceral and neural sclerosis the parenchyma of the cell is primarily at fault, and that connective tissue and vascular proliferations are secondary lesions. Grandmaison furnishes histological proof of this in induced (experimental) hepatic sclerosis. This may be equally true of cerebro-spinal sclerosis, though without definite proof in the present incomplete stage of pathological anatomy. L. F. B.

RECENT RECORDS OF PARESIS.

In the "Gazette médicale de Paris," February 13, 1892, there is a note on Dr. Henri Evard's "Contribution to the Study of the Etiology of General Paralysis," based upon fifty-two carefully recorded cases. Judging from these the author inclines to the belief that the most frequent causes of paresis are, first, a hereditary tendency to congestion and to conditions that predispose to congestions. The French journal calls attention to the fact that Evard neglects to mention lithæmia as a factor, and states that this is the origin of the congestive tendencies that he so readily recognizes. The second and occasional cause of paresis in a predisposed subject, is alcoholism. And the third, active and unmistakable, is syphilis. This view is opposed to that of Charcot, who looks upon both alcoholism and syphilis as exciting causes of paresis in persons predisposed by heredity to vaso-motor and trophic disturbances. The foregoing diseases in the parents may produce the paretic constitution in the child, and in this way induce paresis. Thus can be explained the existence (of which eight cases have been recorded) of general paralysis or diffuse meningo-encephalitis in adolescents. Paresis in women resembles that of children; and is manifested in both instances, psychically, as a primary dementia.

The duration of paresis is considered at some length by Salgó (Centralb. f. Nervenh. u. Psych., 1891), who divides this disease into four different forms, each classified according to the nature of its various stages and the particular course that it runs. The first is the *foydroyant* form that the author identifies with certain cases of rapid and fatal amentia. The second is depressive and hypochondriacal, in which the duration is somewhat like